Benign Paroxysmal Positional Vertigo Accompanied by Sudden Sensorineural Hearing Loss: A Comparative Study With Idiopathic Benign Paroxysmal Positional Vertigo

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Objectives/Hypothesis: To investigate the clinical characteristics of benign paroxysmal positional vertigo (BPPV) associated with idiopathic sudden sensorineural hearing loss (ISSHL) and to compare them with the characteristics of idiopathic BPPV (i-BPPV).

Study Design: Retrospective case series.

Methods: We retrospectively analyzed 519 patients with ISSHL and 597 patients with i-BPPV. The ISSHL patients with recent vertigo history before or after admission were tested with video-nystagmography that included the caloric test. BPPV with same-side ISSHL was identified and categorized as secondary BPPV (s-BPPV) using the roll or Dix-Hallpike test. All members of the s-BPPV and i-BPPV groups underwent a daily canalith repositioning procedure (CRP) during the admission periods. We investigated the clinical characteristics, including the number of CRPs performed to achieve successful reposition, canal involvement type, and effect of canal paresis and made comparisons between the s-BPPV and i-BPPV groups.

Results: Of the 519 ISSHL patients, 63 (12.1%) were identified as having s-BPPV. Multicanal involvement was more frequent in s-BPPV than i-BPPV patients ($P < .001$). The mean number of CRPs needed to achieve successful reposition was 4.28 in s-BPPV and 1.34 in i-BPPV ($P < .001$). The presence of canal paresis was also associated with a greater number of CRPs required for s-BPPV ($P < .02$).

Conclusions: In about 12% of ISSHL patients, s-BPPV was concurrent. More CRPs were required for successful repositioning in patients with s-BPPV than in patients with i-BPPV. Also, the presence of canal paresis in s-BPPV was associated with a greater number of required CRPs.

Key Words: Sudden hearing loss, benign paroxysmal positional vertigo, prognosis, otolith.

Level of Evidence: 4


INTRODUCTION

Secondary benign paroxysmal positional vertigo (s-BPPV) can be caused by various inner ear diseases, including idiopathic sudden sensorineural hearing loss (ISSHL), Meniere’s disease, neurolabyrinthitis, and vestibular neuritis.1–6 Head trauma and surgery have also been reported to be related to development of s-BPPV.7–9 According to the literature, s-BPPV is considered to be more recurrent, have longer disease duration, and have a poorer prognosis than idiopathic BPPV (i-BPPV).3–5 Even so, s-BPPV is underdiagnosed as it is often considered minor compared to the primary disease. However, focusing exclusively on the primary disease and neglecting to properly diagnose and treat s-BPPV can have negative impacts on quality of life in s-BPPV patients.

The etiologies of ISSHL have not been clearly identified, but generally accepted causes are viral infection, vascular compromise, and intracochlear membrane rupture.10–13 In 30% to 40% of ISSHL patients, vertigo is accompanied by hearing loss.14–16 Vertigo accompanied with ISSHL occurs in specific disease conditions, such as benign paroxysmal positional vertigo (BPPV), neurolabyrinthitis, unilateral vestibulopathy, and central vertigo. With any of these conditions, BPPV secondary to ISSHL may develop through involvement of the cochlear and vestibular endolymphatic system via specific etiologic factors, causing detachment of otolith on utricular macula.1,3

The purpose of our study was to investigate the clinical characteristics of BPPV associated with ISSHL and to compare them with those of i-BPPV.

MATERIALS AND METHODS

Subjects

We performed a retrospective analysis of patients who had been diagnosed as having ISSHL or i-BPPV between January 2005 and July 2011 at our tertiary care hospital. This study’s protocol was approved by the institutional review board. All patients underwent a detailed neuro-otologic examination that included clinical history, otoscopy, and bedside vestibular function tests. The ISSHL subjects experienced subjective unilateral sensorineural hearing loss that had developed within 72 hours,
with a minimum 30 dB of hearing loss at three consecutive frequencies in pure-tone audiometry.\textsuperscript{17} The ISSHL patients were treated with proper steroid treatment as inpatients. For ISSHL patients with recent vertigo history before or after admission, video-nystagmography including caloric test was performed. Based on the results of the Dix-Hallpike test and the roll test, BPPV accompanied by same-side ISSHL was diagnosed. We categorized this group as s-BPPV. Next, we searched our hospital inpatient database for BPPV patients with typical BPPV-associated nystagmus but neither inner ear disease nor any other abnormal neuro-otological findings, and these were classified as i-BPPV. The i-BPPV group was also treated by admission as our hospital policy. Both s-BPPV and i-BPPV subjects were excluded if they had Meniere’s disease, central nervous system disorder, trauma, or middle ear disease. Also, bilateral BPPV was excluded because it was not clearly correlated with ISSHL.

### Data Collection

We obtained patient demographic data and clinical data such as the relative timing of hearing loss, vertigo symptoms, and the involved canal type.

Members of the s-BPPV and i-BPPV groups underwent oncedaily canalith repositioning procedures (CRPs) until symptoms and typical nystagmus subsided. Patients with posterior canal BPPV were treated with the Epley canalith repositioning procedure with or without the Brandt-Daroff habituation method.\textsuperscript{16,19} Horizontal canal BPPV patients with canalithiasis were treated with the barbecue rotation maneuver and forced prolonged positioning.\textsuperscript{20} Patients with cupulolithiasis were treated with the same maneuver as used for canalithiasis after conversion of the ototh to canalithiasis by mastoid oscillation or therapeutic head shaking.\textsuperscript{20,21} To compare treatment efficacies in the s-BPPV and i-BPPV groups, the number of CRPs required for successful reposition was examined. If multiple canals were involved, CRP was performed for each of them, but we counted them as only one simultaneous treatment. In ISSHL with vertigo subjects, bithermal air caloric tests were initially performed at temperatures of 50°C and 24°C (Micromedical Technologies Inc., Chatham, IL). The presence of canal paresis (CP) was considered when the interaural difference in nystagmus response (degrees per second of the slow phase velocity) for the two temperatures was 25% or greater using Jongkees’ formula.

### Statistical Analysis

Descriptive statistics and frequencies were determined for the studied variables. The numbers of CRPs required for successful reposition in s-BPPV and i-BPPV patients were compared using the independent \( t \) test. Also, the number of CRPs in s-BPPV patients in the presence or absence of CP were compared using the independent \( t \) test. Involved canal types for s-BPPV and i-BPPV were compared using the \( \chi^2 \) test. Treatment outcomes according to canal involvement in the s-BPPV group were assessed by one-way analysis of variance. The correlation between CP value and the number of CRPs in the s-BPPV group was assessed using the Spearman correlation test. Level of significance was defined as a \( P \) value < .05. Statistical analyses were performed using PASW Statistics version 17.0 (IBM SPSS, Armonk, NY).

### RESULTS

We identified 519 patients with ISSHL, 63 of whom had s-BPPV, and 597 patients with i-BPPV in our hospital BPPV database who met the inclusion criteria were also identified. Table I shows the characteristics of the overall study population. The prevalence rate of s-BPPV in ISSHL was 12.1%. Age and sex distributions did not differ between the s-BPPV and i-BPPV groups, and both were slightly female dominant. Multiple canal involvement was more frequent in the s-BPPV group \((P < .001, \chi^2 \) test; Fig. 1). However, the incidences of other types of canal involvement, such as posterior, lateral, and anterior, were the same in the two groups. In both groups, posterior canal involvement was the most frequent, and anterior canal involvement was the least (Table I).

As shown in Figure 2, the number of CRPs needed for successful reposition was higher in the s-BPPV than in the i-BPPV group. The number of CRPs was 4.28 \pm 2.12 (mean \pm standard deviation) in the s-BPPV group and 1.34 \pm 1.74 in the i-BPPV group. The group

### TABLE I. Characteristics of the Study Populations.

<table>
<thead>
<tr>
<th>Involved Canal Type</th>
<th>s-BPPV (n = 63)</th>
<th>i-BPPV (n = 597)</th>
<th>( P ) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SC</td>
<td>28 (44.4%)</td>
<td>293 (49.8%)</td>
<td>.37</td>
</tr>
<tr>
<td>LS</td>
<td>21 (33.3%)</td>
<td>247 (41.4%)</td>
<td>.06</td>
</tr>
<tr>
<td>ASC</td>
<td>0 (0%)</td>
<td>11 (1.8%)</td>
<td>--</td>
</tr>
<tr>
<td>Multicanal</td>
<td>14 (22.2%)</td>
<td>46 (7.7%)</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

\( * \)Independent \( t \) test.

\( \chi^2 \) test.

s-BPPV = BPPV secondary to ISSHL (idiopathic sudden sensorineural hearing loss); i-BPPV = idiopathic BPPV; M = male; F = female; PSCC = posterior semicircular canal; LS = lateral semicircular canal; ASC = anterior semicircular canal; multicanal = more than two semicircular canals.

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difference was significant ($P < .001$). Because we only checked CP in ISSHL group, we could analyze the effect of CP on the number of CRPs only in the s-BPPV group. In s-BPPV patients, the presence of CP also affected the number of CRPs (Fig. 3), which was $4.73 \pm 3.84$ in the presence of CP and $2.72 \pm 1.76$ in the absence of CP ($P = .02$). However, the CP value was not correlated with the number of CRPs in patients with CP (Fig. 4). As shown in Figure 4, four patients with CP required only one or two CRPs for successful reposition, which meant that not all of the patients required multiple treatments even in the presence of CP. As we analyzed the treatment outcome according to the involved canal, the
treatment outcome was not associated with the involved canal type in s-BPPV (Fig. 5).

Figure 6 shows the relative timing of hearing loss and vertigo symptoms in secondary BPPV. Simultaneous hearing loss and vertigo (68%) were the most common presentation of symptoms in s-BPPV, followed by development of hearing loss (20%), and, least commonly, vertigo (11%). In all cases, the second symptom occurred within 3 days of the first.

DISCUSSION

BPPV is the most common cause of peripheral vertigo. However, the pathophysiology of i-BPPV is not clearly identified. According to Schuknecht, BPPV is caused by otocochlear sudden sensorineural hearing loss. However, some s-BPPV patients developed hearing loss first (20%) or vertigo first (11%), with the second symptom occurring within 3 days of the first.

etiology is rare. However, in our experience a considerable portion of ISSHL with unknown etiology has continuous and progressive clinical features caused by the clinical characteristics of s-BPPV described in the present study. We found the following clinical characteristics of s-BPPV related to ISSHL.

First, s-BPPV secondary to ISSHL required a larger number of CRPs for successful reposition and therefore a longer disease duration than i-BPPV. In our previous study, we found a similar result. If changes of the inner ear environment were consistent during the active disease state, such as viral infection, then otolith detachment would continuously occur until complete recovery of the inner ear environment. Consequently, although successful reposition of the otolith was achieved by the initial CRP, repeated CRPs were necessary. In a recent study, Song et al. reported 8.6% of s-BPPV associated with ISSHL. We believed that it was a reasonable prevalence rate compared with our study. However, they revealed that the recurrence rate of s-BPPV within 1 week was 69.2%. In our opinion, this phenomenon might be considered as incomplete reposition or effect of residual otolith in the canal due to continuous and large amount detachment rather than early recurrence.

Second, presence of CP affected the number of CRPs needed for successful reposition in s-BPPV patients, but CP values by Jongkees’ formula were not correlated with the number of needed CRPs. The presence of CP in ISSHL implied that marked vestibular damage was developed simultaneously with cochlear damage. Regarding s-BPPV development, it could be that a large amount of otocochlear detachment was continuously occurring. However, in the current study higher CP values did not correlate with larger numbers of required CRPs. Moreover, 100% of CP value, which assumes nearly complete loss of vestibular function, was not identified in all s-BPPV cases. For the typical presentation of vertigo and nystagmus in BPPV, inner ear disease detaches the otocochlea but does not totally destroy semicircular function. This may explain why the CP values were not correlated with the number of required CRPs. In other words, with very high CP values, the typical presentation of nystagmus and vertigo related with position change may not be clearly present due to loss of semicircular function.

A third finding in the current study is that the relative timing of hearing loss and vertigo symptoms varied. That is, although the most frequent occurrence of primary symptoms was simultaneous development of hearing loss and vertigo, some cases presented hearing loss first, and others (least frequently) presented vertigo first. If viral neurulabyrinthitis is the cause of the ISSHL, then because it is continuous and progressive, the timing of vestibular system and cochlear system involvement could vary among cases. We believe that if vascular compromise was the etiology, the relative timing of symptoms could only be simultaneous because vascular compromise would probably affect the entire labyrinth due to a single insult of common cochlear artery.
Another interesting finding in the current study is relatively frequent multiple-canal involvement in s-BPPV patients. This could be a natural result of widespread continuous and progressive vestibular system damage, but it is important to consider this presentation during diagnosis and treatment of s-BPPV associated with ISSHL. Balatsouras et al. reported that benign paroxysmal positional vertigo of multiple canals is not rare and presents a clinical challenge; however, accurate diagnosis results in successful treatment comparable with BPPV of a single canal. Therefore, we emphasize that appropriate diagnosis and treatment for multicanal involvement of s-BPPV in ISSHL patients are important for successful outcomes. In fact, as we have shown in Figure 5, treatment outcome did not differ between multicanal and other single-canal involvement.

CONCLUSION

About 12% of ISSHL patients had s-BPPV in the present study. The clinical characteristics of s-BPPV secondary to ISSHL were different from those of i-BPPV. Multicanal involvement was more frequent for s-BPPV. Although simultaneous hearing loss and vertigo were most frequent, hearing loss first and vertigo first were also presented. From a treatment point of view, s-BPPV patients tended to have longer disease duration and need more CRPs than i-BPPV patients. Also, the presence of CP was associated with an increased number of CRPs in patients with s-BPPV secondary to ISSHL.

BIBLIOGRAPHY